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The effect of propofol on plasma membrane ultrastructure in the intact cells. WEIXIANG JIN, ARND PRALLE, Dept. of Physics, University at Buffalo, SUNY — The mechanism of general anesthesia is still unknown. One drug used for human anesthesia, propofol, has been shown to interact with some ligand gated ion-channels, but also easily dissolves in the lipid bilayer and alters fluidity. Which mechanism dominates or even how anesthesia arises are unclear. We study the influence of propofol on plasma membrane (PM) ultrastructure in intact cells. In the PM, transient submicroscopic nanodomains form by interactions between lipid-acyl-chains or lipid head groups, stabilized by cholesterol. In addition, membrane cytoskeleton further regulates the nanodomains, which then regulate signaling. We study transient propofol effects on these domains from low to clinically relevant propofol concentrations by analyzing diffusion of GFP-tagged outer leaflet/inner leaflet membrane proteins. Using bimFCS we measure diffusion on multiple length scales simultaneously. We observe that at low propofol concentrations, the nanodomains trap GPI-mGFP less, consistent with studies showing that propofol decreases the phase transition temperature of membrane derived vesicles. Interestingly, at clinical relevant concentrations of propofol, the nanodomains trap GPI-mGFP more strongly. This is only observed at 37C. By inhibiting myosin activity or actin filaments (de-)polymerization, we find that the activity of actin filaments further alters the behavior of cholesterol nanodomains due to propofol. We compare the effect of propofol and its analog confirming specificity.

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